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LEAD HAZARDS TO FISH, WILDLIFE, AND INVERTEBRATES: A SYNOPTIC REVIEW

by

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Pb conc. in field collections of selected species of flora + fauna. Values are in mg Pb/kg (ppm) fresh weight (FW) or dry weight (DW)

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
California		
Merced	15 DW	
Sacramento	38 DW	
Other	25 DW	
Northern pintail, Anas acuta		
Adult	7 DW	
Immature	6 DW	
Mottled duck, Anas fulvigula		
Adult	48 DW	
Immature	40 DW	
Canvasback		
Adult	17 DW	
Immature	8 DW	
Redhead, Aythya americana		
Adult	26 DW	
Immature	24 DW	
Lesser scaup, Aythya affinis		
Adult	3 DW	
Immature	2 DW	
Black duck, Anas rubripes		
Adult	8 DW	
MAMMALS		
Field mouse, Apodemus sylvaticus		
Near abandoned Pb mine		
Whole body	(9 - 14) DW	Roberts et al. 1978
Kidney	(39 - 46) DW	
Liver	(12 - 13) DW	
Bone	(189 - 352) DW	
Brain	(6 - 13) DW	
Muscle	(7 - 10) DW	
Control area		
Whole body	1 DW	
Kidney	(9 - 13) DW	
Liver	(5 - 8) DW	
Bone	(11 - 21) DW	
Brain	(3 - 4) DW	
Muscle	(5 - 6) DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Short-tailed shrew, *Blarina brevicauda*		
Carcass		
Near metal smelter	109 DW	Beyer et al. 1985
Control site	18 DW	
From area of high traffic levels (>12,000 vehicles/day)		
Total body	18 DW	Getz et al. 1977c
Gut	24 DW	
Spleen	4 DW	
Liver	5 DW	
Lung	17 DW	
Kidney	12 DW	
Femur	67 DW	
Muscle	10 DW	
From area of low traffic levels (<400 vehicles/day)		
Total body	6 DW	
Gut	3 DW	
Spleen	2 DW	
Liver	1 DW	
Lung	8 DW	
Kidney	4 DW	
Femur	12 DW	
Muscle	5 DW	
Cow, *Bos bovis*		
Missouri, hair		
Near Pb smelter		
Fall	94 DW	Dorn et al. 1974
Winter	87 DW	
Spring	96 DW	
Summer	66 DW	
Control area		
Fall	2 DW	
Winter	4 DW	
Spring	2 DW	
Summer	1 DW	
Dung		
Near roadway	10 DW	Robel et al. 1981
Distant site	8 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Dog, Canis familiaris		
Blood		
Healthy	(0.01 - 0.05) FW	NRCC 1973
Pb-poisoned	(0.06 - 0.15) FW	
Big brown bat, Eptesicus fuscus		
Whole, minus GI tract and large embryos		
Males	47 (20-90) FW	Clark 1979
Females	32 (20-56) FW	
Guano	61 DW	
Stomach contents	4 DW	
Horse, Equus caballus		
Near smelter, British Columbia		
Liver	18 FW	Burrows 1981
Kidney	16 FW	
Bone	88 FW	
Near Pb smelter (some deaths), California		
Liver	(15 - 222) FW	Knight and
Kidney	(14 - 80) FW	Burau 1973
Blood	(0.4 - 0.5) FW	
Control areas		
Blood	(0.1 - 0.3) FW	Jenkins 1980
Bank vole, Clethrionomys glareolus		
Whole body		
Near abandoned Pb mine	(16 - 21) DW	Roberts et al. 1978
Control area	(2 - 3) DW	
Chipmunk, Eutamias townsendii		
Hair		
Roadside location	235 DW	Raymond and
Control area	6 DW	Forbes 1975
Prairie vole, Microtus ochrogaster		
Illinois, whole body		
Near heavy traffic	8 DW	Getz et al. 1977b
Control area	3 DW	
Little brown bat, Myotis lucifugus		
Whole	17 (11-29) FW	Clark 1979
Guano	65 DW	
Stomach contents	26 FW	
Bats, Myotis spp., Florida 1981-1983		
Guano	(3 - 6) DW	Clark et al. 1986

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
White-tailed deer, *Odocoileus virginianus*		
Near zinc smelter, Pennsylvania		
Feces	16 (6 - 37) DW	Sileo and Beyer 1985
Bone	9 (4 - 17) DW	
Teeth	6 (3 - 11) DW	
Kidney	2 (1 - 3) DW	
Liver	<2 DW	
Control area, 100 km from smelter		
Feces	8 (4 - 16) DW	
Bone	6 (3 - 11) DW	
Teeth	2 (1 - 4) DW	
Kidney	0.8 (0.5 - 1) DW	
Liver	<0.4 DW	
Muskrat, *Ondatra zibethicus*		
Liver		
Upstream from mine site	0.2 (Max. 0.3) FW	Niethammer
Downstream	0.7 (Max 1.6) FW	et al. 1985
Sheep, *Ovis aries*		
Meat	<0.2 FW	Bunzl and
Liver	<1.5 FW	Kracke 1984
Kidney	<1.1 FW	
Sheep forage		
Grass		
Green	<12 FW	
Old	<33 FW	
Other	<24 FW	
White-footed mouse, *Peromyscus leucopus*		
Carcass		
Near metal smelter	17 DW	Beyer et al. 1985
Control site	7 DW	
Deer mice, *Peromyscus maniculatus*		
From high density traffic area		
Bone	52 DW	Mierau and
Kidney	9 DW	Favara 1975
Liver	3 DW	
Brain	1 DW	
Feces	154 DW	
From low density traffic area		
Bone	5 DW	

Table 5. (Continued)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Kidney	3 DW	
Liver	1 DW	
Brain	0.1 DW	
Feces	7 DW	
Roadside locations		
Brain	(0.6 - 0.8) DW	Jenkins 1980
Liver	(0.9 - 3) DW	
Kidney	(2 - 8) DW	
Bone	(14 - 52) DW	
Hair	235 DW	
Control areas		
Brain	0.1 DW	
Liver	1 DW	
Kidney	3 DW	
Bone	5 DW	
Hair	6 DW	
Illinois, 1982		
Distance from lead battery reclamation plant		
100 m		
Liver	4 FW	Kisseberth et al. 1984
Kidney	13 FW	
Bone	79 FW	
1,000 m		
Liver	1 FW	
Kidney	3 FW	
Bone	2 FW	
Whole, 1978-1979		
Near Cu-Zn mine		
Juveniles	4 FW	Smith and Rongstad 1982
Adults	5 FW	
Control site		
Juveniles	0.5 FW	
Adults	0.7 FW	
Raccoon, Procyon lotor		
Connecticut, Pb-intoxicated		
Liver, kidney	>35 FW	Diters and Nielsen 1978
Commensal rat, Rattus norvegicus		
Houston, Texas, 1978-1979		
Urban		

Table 5. (Concluded)

Taxonomic group, organism, tissue, and other variables	Concentration[a]	Reference[b]
Bone	125 FW	Way and
Kidney	9 FW	Schroder 1982
Stomach contents	31 FW	
Feces	72 FW	
Rural		
Bone	8 FW	
Kidney	3 FW	
Stomach contents	3 FW	
Feces	8 FW	
Roadside mammals, 1976		
Whole, minus GI tract and large embryos		
Short-tailed shrew		
Near highway	26 (6 - 130) FW	Clark 1979
Distant site	2 FW	
Meadow vole, *Microtus pennsylvanicus*		
Near highway	2 (0.2 - 5) FW	
Distant site	<1.4 FW	
White-footed mouse		
Near highway	5 (0.4 - 41) FW	
Distant site	1 (0.3 - 13) FW	
Common shrew, *Sorex araneus*, UK, 1979		
Near roadway		
Liver	17 DW	Chmiel and
Kidney	46 DW	Harrison 1981
Bone	193 DW	
Pelt	10 DW	
Control site		
Liver	<1 DW	
Kidney	9 DW	
Bone	41 DW	
Pelt	3 DW	

[a]Concentrations are listed as mean, (minimum-maximum), and maximum (Max.).

[b]Each reference applies to data in the same row and in the rows that immediately follow for which no reference is indicated.

Whitefish, Coregonus spp., from Pb-contaminated Swedish lakes, showed depressed blood ALAD and blood chemistry derangement when compared to fish from a reference lake--suggesting that Pb affects natural populations of fish in a manner similar to that observed in laboratory studies (Haux et al. 1986).

The significance of organolead residues in aquatic life is unknown, and merits additional research. In Ontario, Canada, about 16% of all fish sampled contained tetraalkyllead compounds, although none were recorded in water, vegetation, or sediments from the collection sites (Chau et al. 1980). Tetramethyllead reportedly was produced from biological and chemical methylation of several inorganic and organic Pb compounds in the aquatic environment, and has been detected at low concentrations in marine mussels, lobsters, and bony fishes (Wong et al. 1981).

AMPHIBIANS AND REPTILES

Tadpoles of bullfrogs (Rana catesbeiana) and green frogs (R. clamitans) from drainages along highways with different daily average traffic volumes (4,272 to 108,800 vehicles per day) contained elevated amounts of Pb (up to 270 mg/kg dry weight), which were positively correlated with Pb in sediments and with average daily traffic volume. Lead in tadpoles living near highways may contribute to the Pb levels reported in wildlife that eat tadpoles. Diets with amounts of Pb similar to those in tadpoles collected near heavily traveled highways have caused adverse physiological and reproductive effects in some species of birds and mammals (Birdsall et al. 1986). Elevated Pb concentrations also were recorded in various species of amphibians and reptiles collected near Pb smelters and mines (Table 5).

BIRDS

In general, Pb concentrations were highest in birds from urban locations (perhaps reflecting greater exposure to automotive and industrial contamination) and in birds near Pb mining and smelting facilities. Lead residues also are greatest in older birds (especially in bone, because of accumulation over time), in sexually mature females, and in waterfowl that have ingested Pb shot pellets (Table 5).

Continued deposition of Pb shot by hunters into wetlands habitats exposes birds to lead. Lead shot is a substantial localized source of contamination, especially in prime waterfowl habitat (Bellrose 1951, 1959; NRCC 1973; White and Stendell 1977; Stendell et al. 1979; Wobeser 1981; Clausen et al. 1982; Longcore et al. 1982; Mudge 1983; Driver and Kendall 1984; Hall and Fisher 1985). Several million hunters are estimated to deposit more than 6,000 metric tons of Pb shot annually into lakes, marshes, and estuaries; this represents about 6,440 pellets per bird bagged. Shot densities as great as 860,000 pellets/ha (2,124,000/acre) have been estimated in some locations (Wobeser 1981), although concentrations of 34,000 to 140,000/ha are more

common (Longcore et al. 1982; Driver and Kendall 1984). For example, Pb shot in bottom sediments from Merrymeeting Bay, Maine, a prime waterfowl staging area, averaged 99,932 shot/ha (274,000/acre), and ranged from 59,541 to 140,324/ha; shot were significantly more numerous in silt than in sand sediments. In general, shot sink more rapidly in soft than in firm substrates, and there is only slight carryover of shot from one season to the next in areas with silt or peat bottoms (Wobeser 1981).

Waterfowl and other birds ingest spent shot during feeding and retain them as grit in the gizzard; the pellets are eroded and soluble Pb is absorbed from the digestive tract. In many species, the ingestion of a single pellet is often fatal. Most deaths, however, go unnoticed and unrecorded. Species such as the mallard and pintail that feed in shallow water by sifting through bottom mud are more likely to encounter shot than are species that feed on submerged vegetation or at the surface (Wobeser 1981). Ingested Pb shot was recorded in 6 of 10 duck species; the frequency was 8.1% in American black ducks sampled in Maine during the hunting seasons of 1976 through 1980 (Longcore et al. 1982). In dry seasons, species that probe for food deep in the sediment are especially susceptible (Hall and Fisher 1985). In England, ingested pellets occurred in 3.2% of the total waterfowl in 16 species examined. Incidences of shot were relatively high (7.1% to 11.8%) in four species (Mudge 1983): greyleg goose (Anser anser), gadwall (Anas strepera), pochard (Aythya ferina), and tufted duck (Aythya fuligula). At least 8,000 mallards in Britain die each winter of Pb toxicosis from ingestion of spent shot (Mudge 1983). It is estimated that about 2.4 million ducks die worldwide of Pb shot poisoning each year--and this estimate does not include population losses resulting from the sublethal effects of Pb (Wobeser 1981). Among larger species of waterfowl, outbreaks of Pb poisoning have been documented in Canada geese, whistling swans (Cygnus columbianus), trumpeter swans, and mute swans (Eskildsen and Grandjean 1984). Lead-poisoned waterfowl tend to seek seclusion and often die in areas of heavy cover; these carcasses are rapidly removed by predators and scavengers, and may result in secondary Pb poisoning, especially among raptors such as the bald eagle (Feierabend and Myers 1984; Reichel et al. 1984). Of 293 bald eagles found dead nationwide between 1978 and 1981, 17 (5.8%) probably died of Pb poisoning after ingesting hunter-killed or hunter-crippled waterfowl containing Pb pellets (Reichel et al. 1984).

The relation between embedded shot and lead toxicosis is unclear. The incidence of embedded shot in various species of waterfowl ranged from 11% to 43% in adults, and 2% to 11% in immatures (Perry and Artmann 1979; Perry and Geissler 1980). Many birds that were struck by shotgun pellets but survived may have died prematurely or been eaten by predators. In one study, the bodies of 23% of adult Atlantic brant (Branta bernicla hrota) that died from starvation in New Jersey in 1977 contained embedded lead shot (Kirby et al. 1983). The effects on survival and fecundity of receiving and carrying relatively high frequencies of embedded shot might be significant, and during years of low adult numbers might have substantial population consequences

(Kirby et al. 1983).

Lead in seeds and invertebrates within rights-of-way of major highways probably is not a hazard to adult ground-foraging songbirds, as judged from experiments with the European starling (Sturnus vulgaris). However, the effects of Pb on survival of fledglings are unknown, although Pb causes reductions in blood hemoglobin, hematocrit, ALAD activity, and brain weight (Grue et al. 1986). In another study, Pb concentrations in feather, carcass, and stomach contents of adult and nestling barn swallows (Hirundo rustica) were greater near a major U.S. highway than in a rural area; however, the number of eggs and nestlings, the body weight of nestlings at 17 days of age, and body weights of adults were similar in the two colonies, suggesting that contamination of roadsides with Pb from automobile emissions is not a major hazard to birds that feed on flying invertebrates (Grue et al. 1984).

Signs of Pb poisoning, i.e., depressed blood ALAD activity or elevated blood Pb levels, were reported for birds near a metal smelter (Beyer et al. 1985), in 17% of canvasbacks from Chesapeake Bay in 1974 (Dieter et al. 1976), and in three species of waders from the Dutch Wadden Sea living in an urban postnuptial moulting area (Goede and de Voogt 1985). The decline in submerged aquatic vegetation in Chesapeake Bay and the later shift in diet of some waterfowl species of Chesapeake Bay from the vegetation (Pb content 2.2 to 18.9 mg/kg dry weight), to the softshell clam Mya arenaria (1.3 to 7.6 mg Pb/kg dry weight), or to other bivalve molluscs (0.8 to 20.4 mg Pb/kg dry weight), probably did not increase dietary Pb burdens in these species (Di Guilio and Scanlon 1985).

The significance of trace amounts of organolead residues in birds is unknown. Trialkyllead seems to concentrate in avian kidney, but contributes less than 5% of the total amount of Pb in kidneys (Johnson et al. 1982).

MAMMALS

The highest body burdens of Pb reported in mammals were near urban areas of dense vehicular traffic, near metal mines and smelters, or near plants that reclaimed storage batteries; concentrations were higher in older organisms, especially in bone and hematopoietic tissues (Table 5; Goldsmith and Scanlon 1977; Way and Schroder 1982). A similar pattern of Pb occurrence and distribution was evident for human populations (Barth et al. 1973).

Diet provides the major pathway for Pb exposure, and amounts in bone are indicative of estimated Pb exposure and metabolism (Chmiel and Harrison 1981). Amounts of whole body Pb and feeding habits of roadside rodents were correlated: body burdens were highest in insectivores such as shrews; intermediate in herbivores, and lowest in granivores (Boggess 1977; Getz et al. 1977c). Food chain biomagnification of Pb, although uncommon in terrestrial communities, may be important for carnivorous marine mammals, such as the California sea lion (Zalophus californianus); accumulations were

ighest in hard tissues, such as bone and teeth, and lowest in soft tissues, uch as fat and muscle (Braham 1973). A similar pattern was observed in the arbor seal, Phoca vitulina (Roberts et al. 1976).

The most sensitive index of Pb intoxication in populations of deer mice as the formation of acid-fast-staining intranuclear inclusion bodies within roximal convoluted tubule cells of kidney; secondary indicators included ecreased body weight, renal edema, reticulocytosis, increased urinary ALA xcretion, and decreased hematocrit (Mierau and Favara 1975). Mierau and avera (1975) concluded that Pb pollution from automobile exhausts has had ittle impact on deer mice, and that severe Pb poisoning is unlikely at raffic densities below 200,000 vehicles per day. Others, however, believe hat Pb emissions from automotive exhausts may pose unnecessary risks to arious species of bats, rodents, and mule deer (Odocoileus hemionus). stimated doses of Pb ingested by the little brown bat (Myotis lucifugus) and ighway populations of shrews and voles equaled or exceeded dosages that have aused death or reproductive impairment in domestic animals; further, mean Pb oncentrations in bats and shrews near highways exceed those reported for mall rodents with Pb-induced renal abnormalities collected from abandoned b-mining sites (Clark 1979). Mule deer from the Rocky Mountain National ark, Colorado, that graze on (heavily contaminated) roadside forage must onsume 1.4% of their daily intake from roadsides before harmful amounts of Pb 3 mg Pb/day) are obtained (Harrison and Dyer 1984); however, this value needs o be verified.

Cows (Bos bovis) adjacent to a Pb battery reclamation plant showed signs f Pb toxicosis, including muscle tremors, blindness, dribbling urine, and rooling. Mice trapped within 400 m of the plant had acid-fast-staining ntranuclear inclusions in renal tubular epithelial cells--a useful diagnostic ndicator of Pb poisoning. A faulty air pollution control system at the plant aused deposition of particulate Pb on the cornfield used for cattle forage, nd was the probable source of the Pb toxicosis in the animals (Kisseberth et l. 1984). Industrial airborne Pb pollution is responsible for contamination f cattle and horses (Equus caballus) within 1,000 m of the source, resulting n elevated blood Pb levels in both species, stillbirths and abortions in attle, and some deaths in horses (Edwards and Clay 1977).

Proximity to the smokestacks of metal smelters is positively associated ith increased levels of Pb in the hair (manes) of horses and in tissues of mall mammals, and is consistent with the results of soil and vegetation nalyses (EPA 1972). Lead concentrations were comparatively high in the hair f older or chronically impaired horses (EPA 1972). However, tissues of hite-tailed deer (Odocoileus virginianus) collected near a zinc smelter did ot contain elevated levels of Pb (Sileo and Beyer 1985). Among small mammals ear a metal smelter, blood ALAD activity was reduced in the white-footed ouse but normal in others, e.g., the short-tailed shrew (Beyer et al. 1985).

No data were available on toxic or sublethal effects of Pb to reptiles nder controlled conditions.

IRDS

Lead poisoning resulting from the ingestion of Pb shotgun pellets has een recognized as a cause of waterfowl deaths since the late 1800's (Wetmore 919; Bellrose 1959). More than a million ducks--especially mallards--and eese die annually from Pb shot poisoning (Clemens et al. 1975). The rincipal cause is the ingestion of spent shot by migrating birds feeding in eavily hunted areas. The pellets are retrieved from the marshy bottoms of hallow and deep water by waterfowl in search of feed and grit. Shot retained n the gizzard is solubilized by a combination of the powerful muscular rinding action and the low pH (2.0 to 3.5) of gizzard contents. The released b is available for absorption, producing weakened birds whose reproductive bilities are reduced and that may starve or fall prey to predators (Clemens t al. 1975). Absorbed lead causes a variety of effects leading to death, ncluding damage to the nervous system, muscular paralysis, inhibition of heme ynthesis, and damage to kidneys and liver (Mudge 1983). Lead poisoning in aterfowl is a debilitating disease in which death follows exposure by an verage of 2 to 3 weeks (Friend 1985). During this time, affected birds lose bility, tend to avoid other birds, and become increasingly susceptible to redation and other causes of mortality. Accordingly, acute large-scale ie-offs of Pb-poisoned waterfowl are uncommon (Friend 1985).

The relation between incidence of Pb shot in waterfowl gizzards and iological effects varies widely, and is probably a function of shot vailability caused by differences in shooting intensity, size of pellets, vailability of grit, firmness of soil and sediments, and depth of surface ater (Street 1983). Also, Pb accumulations and the frequency of avian Pb oxicosis following ingestion of Pb shot are modified by the age and sex of ne bird, geographic location, habitat, and time of year (Finley and Dieter 978; Mudge 1983; Srebocan and Rattner 1988).

The effect of diet on vulnerability to Pb makes interpretation of ublished information on experimental Pb poisoning in waterfowl extremely ifficult (Chasko et al. 1984). For example, many mallards on a diet of corn ie within 10 to 14 days after ingesting a single Pb shot, whereas similar irds on a balanced commercial duck ration appear outwardly normal after ngesting as many as 32 pellets of the same size (Wobeser 1981). Also, ultiple nutritional deficiencies may have additional effects in potentiating ne toxicity of Pb in mallards (Carlson and Nielsen 1985).

Birds of prey may ingest Pb in the form of shot from dead or crippled ame animals, or as biologically incorporated Pb from Pb-poisoned waterfowl, nall roadside mammals, and invertebrates (Stendell 1980; Pattee 1984). Lead oisoning in carnivorous birds has been reported in various species of eagles, ondors, vultures, and falcons, and most--if not all--cases seem to result

from ingestion of Pb shot in food items (Custer et al. 1984). Some raptors ingest many shot in a short time. For example, the stomach of a bald eagle suspected of dying from Pb poisoning contained 75 shot (Jacobson et al. 1977). Results of experimental Pb shot poisoning of bald eagles (Table 7) confirmed results of nationwide monitoring showing that 5.4% of all dead eagles found in 1974-1975 died of Pb poisoning, as evidenced by liver Pb levels of 23 to 38 mg/kg fresh weight (Pattee et al. 1981). Ingestion of food containing biologically incorporated Pb, although contributing to the Pb burden of carnivorous birds, is unlikely in itself to cause clinical Pb poisoning (Custer et al. 1984). A similar case is made for powdered Pb (Franson et al. 1983), and forms of Pb other than shot (Table 7); the strong indication is that the form in which Pb is ingested is crucial.

Signs of Pb poisoning in birds have been extensively documented (Bellrose 1951, 1959; Jordan and Bellrose 1951; Clemens et al. 1975; Forbes and Sanderson 1978; Hunter and Wobeser 1980; Pattee et al. 1981; Wobeser 1981; Franson and Custer 1982; Johnson et al. 1982; Eastin et al. 1983; Kendall and Scanlon 1983; Street 1983; Di Giulio and Scanlon 1984; Fimreite 1984; Gjerstad and Hanssen 1984; Hudson et al. 1984; Anderson and Havera 1985; Burger and Gochfeld 1985; Carlson and Nielsen 1985; Friend 1985; Hoffman et al. 1985a; Lumeij 1985; Beyer et al. 1988). Outwardly, Pb-poisoned birds show the following signs: loss of appetite, lethargy, weakness, emaciation, tremors, drooped wings, green liquid feces, and impaired locomotion, balance, and depth perception. Internally, Pb-poisoned birds show microscopic lesions of the proventricular epithelium, pectoral muscles, brain, proximal tubular epithelium of the kidney, and bone medullary osteocytes; an enlarged bile-filled gall bladder; anemia; elevated protoporphyrin IX levels in blood; decreased ALAD activity levels in blood, brain, and liver; reduced brain weight; abnormal skeletal development; cephalic edema; and esophageal impaction. Postmortem examination of Pb-poisoned birds may show edematous lungs; serous fluid in the pleural cavity; bile regurgitation; abnormal gizzard lining; a usually pale, emaciated, and dehydrated carcass; and elevated Pb levels in liver (>2 mg/kg fresh weight, >10 mg/kg dry weight), kidney (>6 mg/kg dry weight), and blood (> 0.2 mg/l).

Toxic and sublethal effects of Pb and its compounds on birds held under controlled conditions vary widely with species, with age and sex, and with form and dose of administered Pb (Table 7). Several generalizations are possible: decreased blood ALAD and increased protoporphyrin IX activity levels are useful early indicators of Pb exposure; Pb shot and certain organolead compounds are the most toxic forms of Pb; nestlings are more sensitive than older stages; and tissue Pb concentrations and pathology both increase in birds given multiple doses over extended periods (Table 7).

Table 7. Lethal and sublethal effects of lead to selected species of birds.

Species, route of administration, dose, and other variables	Effects	Reference[a]
Northern pintail, Anas acuta		
Single oral dose of 2 No. 5 pellets	No difference from control group in band recovery rate from hunter kills.	1
Mallard, Anas platyrhynchos		
Single oral dose of 1 No. 4 shot (1.4 g)	Some deaths. Residues (mg/kg fresh weight) >3 in brain, >10 in clotted heart blood, >6 in kidney, and up to 20 in liver.	2
Single oral dose		
1 No. 6 shot (1.0 g)	Mortality 9% in 20 days.	3
1 No. 4 shot (1.6 g)	Mortality 19% in 20 days.	3
2 No. 6 shot (2.0 g)	Mortality 23% in 20 days.	3
4 No. 6 shot (4.0 g)	Mortality 36% in 20 days.	3
6 No. 6 shot (6.0 g)	Mortality 50% in 20 days.	3
8 No 6 shot (8.0 g)	Mortality 100% in 20 days.	3
Single oral dose of 1 No. 4 shot (205 mg), equal to 151 mg/kg body weight (BW)	Some deaths; blood ALAD activity depressed 30% after 3 months, 15% after 4 months.	4
Single oral dose of 1 No. 4 Pb shot (200 mg)	Residues (mg/kg dry weight femur) 488 in laying hen, 114 in nonlaying hen, and 9 in drake.	5
Single oral dose of 1 shot (200 mg)	After 30 days, residues (mg/kg fresh weight) 1.0 in blood, 2.5 in liver, and 0.5 in brain. Decrease in ALAD activity in blood and cerebellum.	6

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Single oral dose of shot	Dosed birds recaptured in significantly greater numbers than controls.	7
Single oral dose of tetraethyllead	LD-50 of 107 mg/kg BW. Signs of intoxication included excessive drinking, regurgitation, hypoactivity, muscular incoordination, fluffed feathers, eyelid drooping, tremors, and loss of appetite. Regurgitation within 7 minutes, other signs as soon as 20 minutes, and death usually between 1 and 4 days posttreatment. Remission took up to 8 days.	7a
Fed diets containing 25 mg Pb/kg, as lead nitrate, for 12 weeks	No deaths; no pathology; no significant accumulations of Pb in liver, kidney, or bone; no changes in hemoglobin or hematocrit; decrease in blood ALAD activity, and increase in blood Pb levels--both returned to normal within 3 weeks on Pb-free diet.	8
Fed diets containing 100 mg Pb^{2+}/kg	Elevated levels in bone (9.6 mg/kg fresh weight vs. 0.7 in controls) and egg (1.3 vs. 0.9 in controls).	9
Fed diets containing metallic Pb for 42 days		
100 mg/kg diet dry weight	Elevated Pb levels (mg/kg dry weight) in kidney (23) liver (7), and bone (5).	10

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
10 mg/kg diet	Residues (mg/kg dry weight) of 4 in kidney (vs. <0.5 in controls), 0.7 in liver (vs. <0.5 in controls), and 0.8 in bone (vs. 0.9 in controls).	10
Ducks, *Anas* spp.		
Single oral dose of 2 shot (254 mg) or 5 shot (635 mg)	Weight loss, emaciation, elevated Pb concentrations in bone, some deaths. American black duck, *Anas rubripes*, more sensitive than mallards.	11
Birds		
Dietary route, 11 species, diagnosed as Pb-poisoned	All had inclusions in proximal convoluted tubules of kidney; liver Pb residues ranged from 3.1 to 15 mg/kg fresh weight.	12
Lethal dietary administration of lead acetate, 6 species	Before death, birds were emaciated and showed increases in blood protoporphyrin and decreases in ALAD; renal intranuclear inclusion bodies were present in 83% of all birds that died from Pb poisoning. Median Pb concentrations (mg/kg fresh weight) ranged in the liver from 20 in male red-winged blackbirds (*Agelaius phoeniceus*) to 111 in female northern bobwhites (*Colinus virginianus*), and in the kidney from 22 mg/kg in the blackbird to 190 in the bobwhite.	13

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Rock dove, Columba livia		
Intragastric administration of 6.25 mg Pb (as lead acetate)/kg BW daily for 64 weeks	Anemia, elevation in erythrocyte porphyrin, kidney pathology; residues (mg/kg fresh weight) of 603 in kidney, 501 in bone, 8 in liver, 2 in brain, 4.4 in blood, 0.8 in sciatic nerve, and 0.1 in crop.	14
Intubation of 6.25 mg Pb (as lead acetate)/kg BW, chronic exposure	Interfered with four-step learning sequence; elevated blood Pb levels remained for 5 weeks after Pb exposure.	15
Japanese quail, Coturnix japonica		
Single oral dose of tetraethyllead	LD-50 of 24.6 mg/kg BW.	7a
Fed diets containing different forms of Pb for 5 days		
5,000 mg metallic Pb/kg	No effect on survival or food consumption.	16
5,000 mg Pb (as lead nitrate)/kg	No overt signs of toxicity.	16
5,000 mg Pb (as lead subacetate $C_4H_{10}O_8Pb_3$)/kg	No overt signs of toxicity.	16
2,761/mg Pb (as lead arsenate)/kg	LD-50.	16
Prairie falcon, Falco mexicanus		
Fed shotgun-killed pheasants and ducks	Death, preceded by vomiting, ataxia, blindness, and	

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
	convulsions. Lead shot recovered from stomach; residues (mg/kg dry weight) of 57 in liver and 78 in kidney.	17
nerican kestrel, Falco sparverius		
Fed mallard homogenate containing 16 to 87 (biologically incorporated) mg Pb/kg fresh weight for 60 days.	Residues of 0.4 mg/kg fresh weight in liver and 7.6 mg/kg dry weight in bone.	18
Oral administration of 1 No.9 shot daily for 60 days	Residues (mg/kg fresh weight) of 0.4 in liver and 28.7 in bone.	18
Fed control diet containing 0.4 mg Pb^{2+}/kg fresh weight	Residues of 0.1 mg/kg fresh weight in liver and 4.2 mg/kg dry weight in bone.	18
Fed diets containing 50 mg metallic Pb powder/kg for at least 5 months	Blood ALAD reduced 80%; liver residues of 1.3 to 2.4 mg/kg dry weight; no effects on blood chemistry.	19
As above, except diet contained 10 mg/kg	No measurable effects.	19
Fed diets containing metallic Pb powder for 6 months		
50 mg Pb/kg diet	No adverse effects on survival, egg laying, fertility, or eggshell thickness. Elevated residues (mg/kg dry weight) in humerus (13), tibia (62), and liver (2).	20

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
10 mg Pb/kg diet	Elevated Pb in bone (4 to 9 mg/kg dry weight vs. <0.8 in controls) and in liver (3 vs. <0.5 in controls).	20
Nestlings dosed orally with metallic Pb powder daily for 10 days		
625 mg/kg BW	Mortality (40% in 6 days); reduced growth; reduced kidney and liver weight; abnormal skeletal development; ALAD depression in all tissues examined; elevated burdens (mg/kg fresh weight) in kidney (15), liver (6), and brain (3).	21
125 mg/kg BW	Reduced growth, reduced brain weight, abnormal skeletal development, ALAD depressions in hematopoietic tissues, elevated burdens (mg/kg fresh weight) in kidney (7), and liver (4).	21
25 mg/kg BW	ALAD depression in all tissues examined; burdens (mg/kg fresh weight) elevated in kidney (3) and in liver 1.4).	21
Fed 60 days with homogenized cockerels (Gallus sp.) containing up to 448 mg (biologically incorporated) Pb/kg dry weight	No effect on survival, growth, hemoglobin, hematocrit, and erythrocyte number. Elevated burdens in kidney, liver, femur, brain, and blood.	22

Table 7. (Continued)

ecies, route of administration, se, and other variables	Effects	Reference[a]
icken, *Gallus* sp.		
Fed diets containing 1,850 mg Pb/kg, as lead acetate, for 4 weeks	No deaths or severe clinical hematological effects; growth rate suppressed 47%, blood Pb residues 3.2 to 8.3 mg/l.	23
ld eagle, *Haliaeetus ucocephalus*		
Oral administration of 10 No. 4 shot (2,000 mg)		
Eagles dying 10 to 133 days posttreatment	Residue levels (mg/kg dry weight) 0.9 in muscle, 1.4 in brain, 6 in kidney, 10 in tibia, 10.3 in humerus, 10.4 in femur, and 16.6 in liver. Loss in body weight 16% to 23% at death.	24
Eagle sacrificed at day 133 posttreatment (bird went blind)	Residue levels (mg/kg dry weight) <0.1 in muscle, 2.1 in brain, 3.2 in kidney, 3.4 in liver, and 12.2 to 13.8 in bone.	24
Controls	Residue levels (mg/kg dry weight) <0.1 muscle, 0.1 in brain, 0.4 in liver, 0.5 in kidney, and 4.5 to 6.6 in bone.	24
illow ptarmigan, *Lagopus agopus*		
Single oral dose		
1 No.6 shot (100 mg)	Weight loss of 12% in 15 days; residues of 3.3 mg/kg fresh weight in liver, 56 mg/kg dry weight in tibia.	25,26

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
3 No.6 shot (300 mg)	Some deaths between days 8 and 15 posttreatment, reduced food intake, weight loss, lethargy, diarrhea; residues of 7.3 mg/kg fresh weight liver, 139 dry weight tibia.	25,26
6 No. 6 shot (600 mg)	If shot retained in gizzard, death resulted; residues (mg/kg) 72 fresh weight in liver, 154 dry weight in tibia.	25,26
Controls	Residues (mg/kg) 0.1 fresh weight in liver, 5 dry weight in tibia.	25,26
Raptors, 4 spp.		
Fed rock doves (Columba livia) and brown hares (Lepus europaeus) containing Pb shot for 3 weeks to 6 months	Death preceded by weight loss, convulsions, and inability to fly. Residues (mg/kg dry weight) at death ranged from 57 to 175 in liver, and 34 to 221 in kidney.	27
Common tern, Sterna hirundo		
Single injection of 200 mg Pb^{2+}	Adverse effects on behavior (locomotion, balance, righting response, feeding tasks, behavioral thermoO regulation); most apparent within 5 days postinjection.	28
Ringed turtle-dove, Streptopelia risoria		
Single oral dose of 2 pellets (220 mg)	Blood Pb (mg/l) 4.69 at 24 hours, and 0.14 at 14 days (vs. control values of 0.004 to 0.012 mg/l); blood ALAD depressed from 24 hours through 14 days.	29

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Single oral dose of 4 shot (440 mg)	Mortality 71% at 6 °C in 7 days; nil at 21 °C in 9 days--but some with seizures and kidney histopathology. No spermatozoa in seminiferous tubules. Lead residues elevated in bone, liver, and brain in both groups, but more elevated in cold-stressed group.	30,31
Single oral dose of 4 shot (440 mg)	Testicular damage in adults held at 6 °C or 21 °C; mortality higher in cold-stressed group.	32
Single oral dose of 4 shot (488 mg)	Some deaths. Intranuclear inclusion bodies in cells of kidney proximal convoluted tubules.	12
Single oral dose of 75 mg Pb/kg BW, as lead acetate	Some deaths; kidney damage.	12
Intubation with 75 mg Pb (as lead acetate)/kg BW daily for 7 days	Residues, (mg/kg dry weight) 457 in kidney, 29 in liver, and 12.4 in brain; seizures; depressed blood ALAD activity; blood Pb concentration 311 ug/l.	33
Controls	Concentrations (mg/kg dry weight) 8.2 in kidney, 3.0 in brain, 1.2 in liver; blood Pb concentration 18 ug/l.	33
Drinking water with 100 ug Pb^{2+}/l for 2 weeks before pairing, and throughout a breeding cycle	Reduction in testes weight and spermatozoa number. No effect on egg production or fertility. Bone Pb levels higher than controls especially in females. Significantly higher Pb concentrations in bone, liver,	

Table 7. (Continued)

Species, route of administration, dose, and other variables	Effects	Reference[a]
	and feather in progeny of Pb-treated parents than in controls.	34
European starling, Sturnus vulgaris		
Oral administration (capsule) of triethyllead chloride at 2,000 ug daily (28 mg/kg BW) for 11 days, or until death	Mortality 100% by day 6. Dying birds showed decreased respiration, squatting, fluffed feathers, and abnormal head posture. Average residues (mg/kg fresh weight) 6.0 in bone, 7.3 in brain 19.9 in kidney, 20.0 in muscle, and 40.2 in liver.	35
As above, but dose was 200 ug daily (2.8 mg/kg BW)	No deaths, reduced food consumption. All tissue residues <2.0 mg/kg fresh weight (vs. <0.1 in controls).	35
Oral administration (capsule) of trimethyllead chloride at 2,000 ug daily (28 mg/kg BW) for 11 days, or until death	Mortality 100% by day 6. Signs included impaired balance, tremors, fluffed feathers, uncoordinated feeding movements, weight loss, inability to fly. Residues (mg/kg fresh weight) averaged 4.3 in bone, 11.0 in muscle, 16.7 in brain, 30.2 in kidney, and 82.4 in liver.	35
As above, but dose was 200 ug daily (2.8 mg/kg BW)	No deaths, survivors hyperactive. Average tissue residues (mg/kg fresh weight) 0.4 in bone, 3.1 in muscle, 3.5 in brain, 3.7 in liver, and 5.4 in kidney.	35

Table 7. (Concluded)

Species, route of administration, dose, and other variables	Effects	Reference[a]
Mourning dove, Zenaida macroura		
Single oral dose		
1 No.8 shot (72 mg)	Mortality 24% in 4 weeks; normal courtship and reproductive activities, but egg hatching significantly reduced; Pb residues elevated in kidney, liver, and bone.	36
2 No.8 shot (144 mg)	Mortality 60% in 4 weeks.	36
4 No.8 shot (288 mg)	Mortality 52% in 4 weeks.	36
Single oral dose of 4 No. 8 shot		
4 days posttreatment	Residues (mg/kg dry weight) 345 to 639 in kidney and 58 to 215 in liver (vs. <12 in controls).	37
8 days posttreatment	Residues (mg/kg dry weight) 1,279 to 1,901 in kidney and 179 to 267 in liver.	37

[a]References: 1, Deuel 1985; 2, Longcore et al. 1974a; 3, Longcore et al. 1974b; 4, Dieter and Finley 1978; 5, Finley and Dieter 1978; 6, Dieter and Finley 1979; 7, Bellrose 1951; 7a, Hudson et al. 1984; 8, Finley et al. 1976; 9, Haegele et al. 1974; 10, Di Giulio and Scanlon 1984; 11, Chasko et al. 1984; 12, Kendall and Scanlon 1985; 13, Beyer et al. 1988; 14, Anders et al. 1982; 15, Dietz et al. 1979; 16, Hill and Camardese 1986; 17, Redig et al. 1980; 18, Stendell 1980; 19, Franson et al. 1983; 20, Pattee 1984; 21, Hoffman et al. 1984a,b; 22, Custer et al. 1984; 23, Franson and Custer 1982; 24, Pattee et al. 1981; 25, Gjerstad and Hanssen 1984; 26, Fimreite 1984; 27, Macdonald et al. 1983; 28, Burger and Gochfeld 1985; 29, Kendall et al. 1982; 30, Kendall et al. 1981; 31, Kendall and Scanlon 1984; 32, Veit et al. 1983; 33, Kendall and Scanlon 1982; 34, Kendall and Scanlon 1981; 35, Osborn et al. 1983; 36, Buerger et al. 1986; 37, Kendall and Scanlon 1983.

Trialkyllead salts are 10 to 100X more toxic to birds than are inorganic Pb salts; they tend to accumulate in lipophilic soft tissues in the yolk and developing embryo, and have high potential as neurotoxicants (Forsyth et al. 1985); accordingly more research is needed on alkyllead toxicokinetics. Some alkyllead compounds have been implicated in bird kills. In autumn 1979, about 2,400 birds of many species were found dead or disabled on the Mersey estuary, England, an important waterfowl and marsh bird wintering area; smaller kills were observed in 1980 and 1981 (Bull et al. 1983). Affected birds contained elevated Pb concentrations in liver (>7.5 mg/kg fresh weight), mostly as organolead. Bull et al. (1983) suggested that trialkyllead compounds were discharged from a petrochemical factory producing alkylleads, into the estuary where they were accumulated (up to 1.0 mg/kg fresh weight) by clams (Macoma balthica) and other invertebrates on which the birds could feed. Birds dosed experimentally with trialkyllead compounds died with the same behavioral and internal signs found in Mersey casualties; tissue levels of trialkyllead were similar in the two groups of birds (Osborn et al. 1983). Sublethal effects that might influence survival in the wild were found in both sublethally dosed and apparently healthy wild birds when tissue levels of trialkyllead compounds were matched in the two groups of birds. It was concluded that trialkyllead compounds were the main cause of the observed mortalities and that many apparently healthy birds were still at risk (Osborn et al. 1983).

Nestlings of altricial species (those confined to the nest for some time after hatch) may be considerably more sensitive to Pb exposure than adults, and also more sensitive than hatchlings of many precocial species (Hoffman et al. 1985a). Hatchlings of precocial species, including chickens, Japanese quail (Coturnix coturnix), mallards, and pheasants, are relatively tolerant to moderate Pb exposure, i.e., there was no effect on growth at dietary levels of 500 mg Pb/kg, or survival at 2,000 mg Pb/kg (Hoffman et al. 1985a,b).

Some species of domestic birds are resistant to Pb toxicosis. for example, blood Pb levels of 3.2 to 3.8 mg/l in Pb-stressed cockerels (Gallus sp.) were much higher than residues considered diagnostic for Pb poisoning in most domestic mammals, except swine--which tolerated up to 143 mg Pb/l blood (Franson and Custer 1982).

MAMMALS

Three stages of recognizable Pb poisoning, or plumbism, have been reported in humans (NRCC 1973): (1) mild or severe dysfunction of the alimentary tract as shown by loss of appetite, constipation, abdominal cramps, headaches, general weakness, and fatigue; (2) atrophy of forearm extensor muscles, or paralysis of these muscles and more striking atrophy; and (3) lead encephalopathy, which occurs frequently in Pb-poisoned infants and young children, but only rarely in industrial workers. In general, people with hepatitis, anemia, and nervous disorders were more susceptible to Pb poisoning (Barth et al. 1973). The transfer of Pb across the human placenta and its potential threat to the fetus have been recognized for more than 100 years; women occupationally exposed to Pb showed a comparatively high abortion rate (Tachon et al. 1983). Sensitivity of the brain to the toxic effects of Pb is

considerably greater in the fetus than in the infant or young child (EPA 1980). Lead is not considered carcinogenic to humans (Tsuchiya 1979). However, reports of chromosomal aberrations in human blood lymphocytes (Barth et al. 1973) suggested that Pb is a probable mutagen.

Signs of plumbism in domestic and laboratory animals (data on feral mammals are noticeably lacking), which are similar to those in humans, have been well documented (Barth et al. 1973; NRCC 1973; Mierau and Favara 1975; Davies et al. 1976; Roberts et al. 1976; Forbes and Sanderson 1978; Nriagu 1978b; Osweiler and Van Gelder 1978; Tsuchiya 1979; Ward and Brooks 1979; EPA 1980; Mahaffey et al. 1980; Hamir 1981; Harrison and Laxen 1981; Burrows and Borchard 1982; Demayo et al. 1982; Hamir et al. 1982; Mykkanen et al. 1982; Tachon et al. 1983; Gietzen and Wooley 1984; Berglind et al. 1985; Table 8). There is general agreement on several details: significant differences occur between species in response to Pb insult; effects of lead are more pronounced with organolead than inorganic lead compounds; younger developmental stages are the most sensitive; and the effects are exacerbated by elevated temperatures, and by diets deficient in minerals, fats, and proteins. Tetramethyllead, for example, is about 7X more toxic than tetraethyllead to animals, and both compounds showed toxic effects earlier than did inorganic Pb compounds. In severe cases, death is usually preceded by impairment of normal functions of the central nervous system, the gastrointestinal tract, and the muscular and hematopoietic systems. Signs include vomiting, lassitude, loss of appetite, uncoordinated body movements, convulsions, stupor, and coma. In nonfatal cases, signs may include depression, anorexia, colic, disturbed sleep patterns, diarrhea, anemia, visual impairment, blindness, susceptibility to bacterial infections, excessive salivation, eye blinking, renal malfunction, peripheral nerve diseases affecting the motor nerves of the extremities, reduced growth, reduced life span, abnormal social behavior, and learning impairment. Lead crosses the placenta and is passed in milk, producing early intoxication of the fetus during pregnancy and the newborn during lactation. High Pb doses in mammals induce abortion, reduce or terminate pregnancy, or can result in stillbirths or an increase in skeletal malformations. These signs, together with Pb levels in blood and tissues and histopathological examination, are used to diagnose Pb poisoning.

Lead adversely affected the survival of sensitive mammals tested at different concentrations (Table 8): 5 to 108 mg Pb/kg BW in rats (acute oral), 0.32 mg Pb/kg BW daily in dogs (chronic oral), and 1.7 mg Pb/kg diet in horses (chronic dietary). Adverse sublethal effects (Table 8) were noted in monkeys given 0.1 mg Pb/kg BW daily (impaired learning 2 years postadministration) or fed diets containing 0.5 mg Pb/kg (abnormal social behavior); in rabbits given >0.005 mg Pb/kg BW (reduced blood ALAD activity) or 0.03 mg Pb/kg BW (elevated blood Pb levels); in mice at 0.05 mg Pb/kg BW (reduced ALAD activity); or in sheep at 0.05 mg Pb/kg BW (tissue accumulations).

Table 8. Lethal and sublethal effects of lead to selected species of mammals.

Species, dose, and other variables	Effects	Reference[a]
Cattle, cows, Bos spp.		
Tissue Pb (mg/kg fresh weight) 0.81 in blood, 26.4 in liver, 50.3 in kidney, and 400 in rumen contents	Signs of clinical Pb toxicosis observed.	1
Calves given 2.7 mg Pb/kg body weight (BW), as Pb acetate, for 20 days; milk diet	Death.	2
Calves given 3.0 to 3.5 mg Pb/kg BW daily for 3 months; grain and hay diet	No effect.	2
Calves given 5 mg Pb/kg BW, as Pb acetate, for 7 days; grain and hay diet	Appeared normal.	3
Calves given 5 mg Pb/kg BW, as Pb acetate, for 7 days; milk diet	Signs of Pb poisoning; some deaths.	3,4
Calves given 5 mg Pb/kg BW daily for 10 to 20 days	Blindness, 16% mortality.	4
Calves given forage containing 5 to 6 mg Pb/kg	Fatal in 2 months.	1
Calves given 5 to 6 mg Pb/kg BW daily for 3 years	Chronic toxicity.	2
Adults given 6 mg Pb/kg BW daily for 3 years	No deaths.	5
Calves given 6 to 7 mg Pb/kg BW daily for 2 months	Fatal.	2

Table 8. (Continued)

Species, dose, and other variables	Effects	Reference[a]
Fed 6 to 7 mg Pb (as Pb acetate)/kg BW daily.	Intoxication within 8 weeks; most dead at day 105.	6
Consumed vegetation (17 to 20 mg Pb/kg fresh weight) near Pb battery recycling plant	Some deaths, mostly younger animals; neurological signs. Lead levels, in mg/kg fresh weight, were 13.8 to 35.8 in blood, 6.9 to 96.5 in feces, 97 in liver, and 138 in kidney. Histopathology of liver and kidney.	7
Calves given 20 mg Pb/kg BW daily	Fatal in 8 to 22 days.	2
Accidentally exposed for 10 days to toxic levels of Pb, as Pb shot, through corn silage. Silage storage area received shot from a nearby trap shooting range. Silage contained 32 mg Pb/kg	1.5% dead (2/70), 27% with signs of poisoning (kidney pathology, hemorrhaging). Tissue Pb concentrations of 16 mg/kg fresh weight in liver, > 32 in kidney, and up to 0.8 in blood.	6
Calves, single oral dose of 220 to 400 mg Pb/kg BW, as Pb acetate	LD-50.	2
Total dose of 50 to 100 grams	Toxic.	6
og, Canis familiaris		
Fed 0.32 mg Pb/kg BW daily	Chronic toxic level.	4
Fed 3 mg Pb/kg BW daily, as lead carbonate	Anorexia and convulsions at 180 days.	8
Fed low calcium/phosphorus diet containing 100 mg Pb/kg,	At 12 weeks, anemia, weight loss, and renal necrosis.	

Although Pb is undeniably toxic at high levels of exposure, the implications of lower levels of exposure are poorly defined (Nriagu 1978b). Behavioral effects such as hyperactivity, distractability, and decreased learning ability, as well as certain peripheral neuropathies, have been ascribed to subclinical Pb exposure (Hejtmancik et al. 1982). Impaired learning ability of Pb-stressed animals showing no obvious signs of Pb intoxication has been documented for rats (Cory-Slechta et al. 1981, 1983, 1985; Angell and Weiss 1982; Nation et al. 1982; Geist et al. 1985; Massaro et al. 1986), sheep (Nriagu 1978b; EPA 1980), and primates (Rice 1985)--although variablity was great in all studies. Some learning deficits may be reversible and may not persist beyond a period of rehabilitation (Geist et al. 1985), and some may be induced only at relatively high exposure levels (Hastings et al. 1984). Abnormal social behavior (usually aggression) has been reported in baboons and monkeys (Hopkins 1970; Nriagu 1978b), although mice showed inhibited development of isolation-induced aggression (Ogilvie and Martin 1982). Altered parent-child relationships were suggested when suckling rats were used as surrogates. In that study, pregnant rats fed diets containing powdered Pb nursed for longer periods than normal, and the resultant offspring were slower to explore their environment (Barrett and Livesey 1983). Lead-exposed pups, with blood Pb levels as low as 200 ug/l (considered elevated but within the "normal" range) at weaning, showed an altered dam-pup interaction that resulted in the dam spending longer periods in the nest than usual. Retarded development of Pb-treated pups may account for the longer bouts of nesting by Pb-stressed dams, and the delay in age at which pups explore and learn. Barrett and Livesey (1983) concluded that maternal behavior was related to delays in pup development, and that the functional isolation of pups from their environment may be the antecedent to altered behavior later in maturity.

No data are currently available on effects of Pb-induced altered parent-offspring relationships, impaired learning ability, or abnormal social behavior for any population of free-ranging wildlife.

Ingestion of Pb-containing paint from bars or walls is a significant cause of death among captive wild animals--including many species of apes, monkeys, bears, ferrets, pinnipeds, foxes, panthers, bats, raccoons, and armadillos--and is probably underreported (Hopkins 1970; Zook et al. 1972; Fowler 1975; Forbes and Sanderson 1978). A similar situation exists for domestic animals--including dogs, cats, goats, horses, swine, cattle, and sheep (Dollahite et al. 1978; Forbes and Sanderson 1978; Osweiler and Van Gelder 1978; Hamir 1981). Passage of laws regulating the amount of Pb in paint has decreased the frequency of Pb poisoning, but many animals are still at risk from this source. Lead also occurs in used motor oils, gasoline, batteries, shot, putty, golf balls, linoleum, and printers ink--all of which are considered sources of Pb poisoning to domestic animals (Dollahite et al. 1978).

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